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Sources of greater fetal vulnerability to airborne polycyclic aromatic hydrocarbons among African Americans

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Abstract

Background—This study attempted to clarify the household and mother's lifestyle factors that contribute to the greater fetal vulnerability of African-American individuals to airborne polycyclic aromatic hydrocarbons (PAH).

Methods—Non-smoking pregnant women with no known risks of adverse birth outcome were monitored for their personal exposure to airborne PAH. Birth outcomes were collected from the hospital medical record. Modification of the airborne PAH effects was statistically examined. In linear regression analyses, modification of PAH effect by demographic, socioeconomic and behavioural traits on birth weight and fetal growth ratio were respectively tested, adjusting for the gestational age, gender, parity, delivery season, maternal body mass index and weight gained during the present pregnancy.

Results—Maternal obesity exacerbated the airborne PAH risk by −491 g per 25th to 80th percentile unit exposure (95% CI −197 to −786 g; $p < 0.01$) among African Americans. In addition, frequent dietary intake of smoked, grilled or barbequed items independently reduced the birth weight of African-American newborns by −204 g (95% CI −21 to −387 g; $p = 0.03$).

Conclusion—Maternal obesity significantly exacerbated the risk of prenatal PAH exposure in African-American newborns. Also, frequent dietary consumption of PAH-laden food items posed an independent risk on the reduced birth weight among African Americans.

Socioeconomically disadvantaged and/or ethnic minorities face greater risks of adverse birth outcomes following prenatal exposure to ambient air pollution.^{1–5} Greater vulnerability to air pollution in such subgroups has been speculated to be related to differences in underlying health status, poorer healthcare access, dietary intake of items with lower nutritional value, co-exposure to other environmental pollutants, social or psychological stress and instability in residential setting.³ However, the extent to which specific individual attributes or neighbourhood-level socioeconomic indicators confound or modify the harms of ambient air

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Competing interests None.

Patient consent Obtained.

Ethics approval This study was conducted with the approval of the the Institutional Review Board of the New York Presbyterian Medical Center.

Contributors HC conducted all statistical analyses and drafted the entire manuscript. FPP designed and led the prospective birth cohort study, contributed to the analysis and interpretation of results, and critically revised the paper.

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pollution remains unknown.³ As a result, identification of the modifiers of ambient air pollution effect, as well as clarification of the mechanisms of their actions represent an urgent research need.³

In our ongoing prospective birth cohort study in New York City, prenatal exposure to airborne polycyclic aromatic hydrocarbons (PAH) significantly increased the risks of lower birth weight,⁶ small for gestational age⁷ as well as preterm delivery⁷ among African Americans. However, similar risks were not observed among Dominican individuals residing in the same neighbourhood. The socioeconomic background of all women in the study was overall homogeneous. The self-reported mean annual household income was US \$20 000 or less for 67% of African Americans and 72% of Dominicans.⁸ Maternal educational attainment was also similar.⁸ Furthermore, mean prenatal exposure levels of airborne PAH were comparable between the two groups.^{7, 8} However, the two groups, based on the self-identified ethnic backgrounds, differ in other co-exposure characteristics,⁹ dietary habits and cultural heritage.⁸ For example, prenatal exposure to second-hand smoke was more intensive among African Americans than Dominicans in our cohort.⁹ Prenatal exposure to second-hand smoke was associated with significantly reduced mental development index at 2 years of age regardless of maternal ethnic background.⁹ The risk of neurodevelopmental delay was exacerbated among those children whose mothers experienced difficulty paying for monthly rent, food or clothing (defined as material hardship) during their pregnancy.⁹

Based on this observation, we posited that lifestyle practices and stress experienced by the mother during her pregnancy contributes to greater risks of airborne PAH among African Americans. In particular, we tested modification of the airborne PAH effect on birth weight by mother's inability to pay for monthly rent, food or clothing (coded as a single variable), maternal prepregnancy obesity, frequency of consuming grilled, barbecued or blackened food (sources of PAH), being unmarried and co-exposure to second-hand smoke more than 2 h a day.

METHODS

A complete description of the study appears elsewhere.^{6, 7} In an ongoing prospective birth cohort study, non-smoking, low-income, African-American and Dominican women with no known risks of adverse pregnancy outcome (eg, inadequate prenatal care, diabetes, hypertension, HIV, or illicit drug use) were recruited from local prenatal care clinics between January 1998 and June 2005. The Institutional Review Board of the New York Presbyterian Medical Center approved the study; and informed consent was obtained from all study participants.

During the third trimester, each woman (N=615) was interviewed and completed a questionnaire on her health, lifestyle and exposure. Following the interview, each was provided with a personal air monitor. It operated for a consecutive 48-h period. Particulate and semi-volatile PAH were analysed for pyrene and eight carcinogenic PAH: benz(a)anthracene, chrysene/isochrysene, benzo(b) fluoranthene, benzo(k) fluoranthene, benzo(a)pyrene (BaP), indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene and benzo(g,h,i)perylene.

Outcomes of interest

Birth weight, coded as a continuous variable (g unit) was the main outcome variable. The birth weight has been validated.^{7, 8} Fetal growth ratio, which is a clinically validated marker of intrauterine growth restriction, was calculated for each newborn as a secondary outcome.^{7, 8} The fetal growth ratio was defined as observed birth weight/mean birth weight

at a given gestational age for each gender and ethnic group based on the 1994–6 US birth weight distribution.¹⁰ The ratio indicates the percentage underweight relative to the mean. The sum of the eight carcinogenic PAH was the main exposure variable. Following natural log (ln) transformation, the variable conformed to the normality. Our exposure unit of interest, a ln unit PAH exposure, was equivalent to an increase from the 25th to the 80th percentile (1.55–4.31 ng/m³) in the overall cohort.

Statistical analysis

Descriptive analysis—The internal validity of the material hardship has been demonstrated in our cohort.⁹ Separate analysis was conducted for each ethnic groups because we previously demonstrated significantly different PAH effects.^{6–9} We considered the following variables for their potential PAH modifying role: being unmarried during the present pregnancy, maternal prepregnancy body mass index (BMI) 30 or greater, any material hardship (ie, inability to pay for food, monthly rent, or clothing during the present pregnancy), second-hand smoke exposure for 2 h a day or more, and consuming smoked, grilled, barbecued items more than twice a week based on our previous analyses and the literature review. We could not examine other correlates of socioeconomic status (ie, degree of maternal education attainment and degree of acculturation) due to their high correlation with ethnic identity in this cohort. We compared the arithmetic mean birth weight (95% CI), considering their normal distribution, per increasing quartile of exposure categories of the airborne PAH.

Stratified linear regression analysis—Separate linear regression analysis was conducted for each ethnic group, further stratified according to the suspected effect modifiers. We controlled for the newborn gender, centered gestational age, square term of the centered gestational age, delivery season, parity, maternal BMI and weight gained during the present pregnancy, to be consistent with our previous analysis.⁷ The sizes of the outcome (ie, birth weight reduction per 25th–80th percentile increase in exposure) were compared between the modifier-stratified groups. Reference PAH effect was defined as birth weight reduction in the adjusted PAH regression model for each ethnic group.

In a comprehensive model, each interaction term was forward selected to the baseline model. The baseline model was adjusted for the above confounders and PAH effect modification by maternal ethnic background (ie, airborne PAH×African-American).⁶ The given interaction term was retained in the comprehensive model if the $-2 \log$ likelihood value was significantly larger under the χ^2 distributions with $\alpha=0.05$ than the model without the interaction term. If the interaction term under consideration induced notable multicollinearity (variance inflation factor ≥ 30), the term was eliminated.¹¹ In the final model, the highest variance inflation factor was 9.15, suggesting stability in the estimated size of birth weight reduction. Similar linear regression on the fetal growth ratio per same unit exposure was conducted controlling for the same set of confounders as above.

RESULTS

Exposure and lifestyle characteristics of the maternal cohort

African-American pregnant women were more likely to report social disadvantages and co-exposures during pregnancy than Dominican women (reference group) (table 1). The African-American women reported more intensive daily (h/day and number of cigarette smoked in the woman's presence/day) and chronic (months/gestation) second-hand smoke exposure not only during the present pregnancy, but also within the past 2 years (h/day and months/gestation), compared with the Dominican women (see table 1). Prevalences of being unmarried during the current pregnancy, obesity and the frequency of the dietary intake of

grilled, barbecued or blackened food items were significantly higher among African-American women than Dominican women. On the other hand, a higher proportion of the African-American women (68%) reported that they did not experience any material hardship, compared with the Dominican women (54%).

Modification of PAH effect on birth weight

With increasing prenatal PAH exposure quartile, mean birth weight reduction was significantly reduced for the African-American newborns (table 2). In particular, the size of the reduction at the highest quartile range was larger for the African Americans whose mothers had the following respective characteristics: unmarried; exposed to second-hand smoke 2 h a day or more; or frequently consumed dietary PAH. Reference groups were the African-American newborns at the highest quartile range without the corresponding traits.

Based on the multivariate linear regression models, the estimated birth weight reduction per 25th–80th percentile increase in airborne PAH was 10-fold greater for those from obese mothers than those from non-obese mothers only among the African Americans (see figure 1). The African-American newborns of either unmarried mothers or those who experienced material hardship did not show considerably greater reduction than that in the overall African Americans (see figure 1). The African-American newborns whose mothers experienced second-hand smoke exposure (≥ 2 h/day) had a somewhat larger PAH effect (–200 g, 95% CI 67 to –468 g) compared with those from less than 2 h a day of exposure (–117 g, 95% CI 1 to –235 g) (see figure 1). The sizes of the airborne PAH effects were somewhat larger for the African-American newborns whose mothers frequently consumed dietary PAH sources (–183 g, 95% CI 17 to –383 g) than those who did not (–84 g, 95% CI 55 to –224 g) (see figure 1).

In the final comprehensive model (see table 3, model 1), a 25th–80th percentile increase in airborne PAH exposure was associated with a –164 g greater birth weight reduction for the African-American newborns (95% CI –51 to –277 g; $p < 0.01$) than the Dominican newborns when putative effect modifiers were not considered. However, the same unit exposure was associated with a –491 g greater reduction of birth weight (95% CI –197 to –786 g; $p < 0.01$) for those newborns of obese African-American mothers than those from non-obese Dominican mothers (table 2, model 2). Frequent consumption of grilled or barbecued items among the African Americans was associated with an additional reduction of –204 g (95% CI –21 to –387 g) in birth weight per unit exposure (table 2, model 3).

Modification of PAH effect on fetal growth ratio

Furthermore, a one unit increase in airborne PAH exposure yielded a 14.5% greater reduction of the fetal growth ratio for those born to obese African-American mothers (95% CI –5.7 to –23.2%; $p = 0.001$) compared with those from non-obese Dominican mothers. Frequent dietary intake of grilled or barbecued food among the African Americans was associated with an additional reduction of 6.0% in the fetal growth ratio (95% CI –0.4 to –11.3%; $p = 0.04$).

DISCUSSION

In this group of low-income, non-smoking, young pregnant women, we examined individual-level modifiers of the effect of PAH air pollution on birth weight and the fetal growth ratio, respectively. Among the African Americans, prepregnancy obesity and frequent consumption of PAH-containing foods, respectively, modified the airborne PAH risk, compared with the same unit exposure in Dominicans who lack either trait. Other

characteristics (ie, absence of spousal support during the present pregnancy, second-hand smoke exposure ≥ 2 h/day and material hardship) did not modify the airborne PAH risk.

The observed sizes of PAH effect modifications here require careful interpretation. This is because the estimated sizes of PAH effects on both birth weight and fetal growth ratio reductions depend not only on the maternal characteristics, but also on the scale and reference point of the outcome. For example, clinical consequences of a 5% reduction in fetal growth ratio are expected to be much more serious for a newborn with moderate growth restriction status (75–79.99) than a healthy newborn (≥ 85). Our results thus suggest that the PAH effects depends not only on maternal traits and behaviours, but also on the expected growth potential of the fetus.

Several limitations of the present analysis need to be noted. First, a higher prevalence of multiple behavioural disadvantages and environmental co-exposures among the African-American women suggest that the threat of residual confounding exists. To address this, we chose models with a low to moderate variance inflation factor (<10), in which the effect size for the interaction terms remained stable. Variance inflation factors of the models shown in table 3 indicated that the models are robust. Second, our questionnaire for the pregnant women focused on the individual-level characteristics during the current pregnancy, but other household characteristics, cumulative effects of other chronic stress, preclinical markers of compromised health conditions and their interactions with neighbourhood characteristics (eg, social isolation, neighbourhood crime and poverty index) might also be important predictors of the birth outcomes. These were not examined here.

Strengths of the current study include strict recruitment criteria, which mitigated the likelihood of individual-level confounding. Also, we observed a consistent dose-related reduction for both birth weight and the fetal growth ratio, a validated marker of intrauterine growth restriction, among obese African Americans.

The mechanism of greater PAH susceptibility among obese mothers requires clarification. Previous epidemiological observations have shown that B α P, a lipophilic pro-carcinogen, bioaccumulates in the mammary glands and adipose tissues.¹² Within a murine model, other PAH readily accumulated in lipid droplets of the alveolar macrophages and adipocytes following inhalation.¹³ In the women who have prepregnancy obesity, intensive lipolysis and lipogenesis during pregnancy induce considerable mobilisation of the sequestered B α P along with free fatty acids.¹² Therefore, greater bio-availability of the released PAH to the embryonic/fetal systems during placentation and/or pregnancy maintenance might account for the greater adverse effect of the airborne PAH among obese women. Alternatively, PAH might influence the endocrine function of the adipose tissues directly.¹² For example, chronic administration of B α P in mice inhibited catecholamine-induced adipose tissue lipolysis.¹² Such mice underwent significant weight gain.¹²

Consistent with our earlier observations,^{6–8} prenatal PAH exposure posed no risk to the fetal growth of the Dominican newborns. Such a trend is consistent with the paradoxical birth outcomes among Mexican immigrants living in the USA. Despite their high-risk demographic and socioeconomic profile, Mexican-American newborns had birth outcomes that are comparable to those of US non-Hispanic whites with adequate prenatal care.¹⁴ The apparent lack of association between airborne PAH and adverse birth outcomes in Dominican newborns might reflect healthful cultural practices among recent Dominican immigrants, such as the consumption of food with high nutritional value and close social support.

Our observations suggest possibly complex roles of maternal obesity and dietary PAH intake in heightened susceptibility to airborne PAH among of African Americans. In the US

population, the ingestion of PAH through grilled, blackened or barbecued food constitutes a major route of exposure.¹⁵ Considering the importance of suboptimal fetal growth in life-course morbidity and mortality, future studies are needed to confirm the potential endocrine-disrupting properties of the PAH in a larger sample.

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What is already known on this subject

- PAH are globally generated and distributed mutagens and pro-carcinogens, emitted during incomplete combustion and/or pyrolysis of solid fuel, including industrial or domestic coal, wood, cigarette and food items.
- Prenatal exposure to PAH significantly elevates the risks of preterm delivery and intrauterine growth restriction in African Americans.
- Aetiologies underlying the greater vulnerability of African Americans to prenatal PAH exposure remains unknown.

What this study adds

- Prenatal airborne PAH exposure for those born to obese African women was associated with significantly greater reduction in the birth weight and fetal growth ratio, respectively.
- Both ambient exposure to PAH and high dietary intake of grilled or barbecued meat or fish during pregnancy contributed to a significantly lower birth weight of African Americans.
- Inhaled or ingested PAH might interfere with the endocrine function of the mother–fetus pair.

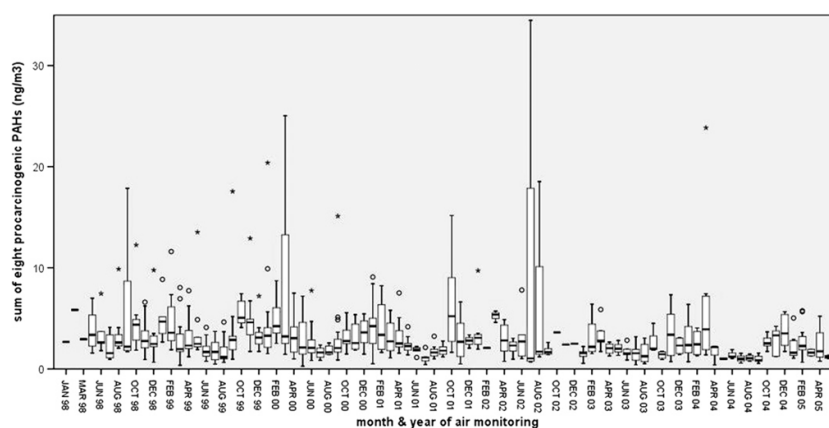


Figure 1. Distribution of summed eight pro-carcinogenic polycyclic aromatic hydrocarbons (PAH) (ng/m^3) over the air monitoring period. The box and the line within it represent the 25th, the median and the 75th percentile values, respectively. The symbols \circ and $*$ represent observations that are 1.5-fold and greater and threefold larger, respectively, than the interquartile range.

Table 1

Demographic and exposure characteristics of the cohort

	African Americans (N=224)		Dominicans (N=391)		p Value*
	N (%)	Mean±SD	N (%)	Mean±SD	
Prepregnancy BMI					<0.01
Underweight (<20 kg/m ²)	31 (14%)		58 (16%)		
Appropriate (20–24.9 kg/m ²)	66 (31%)		167 (46%)		
Overweight (25–29.9 kg/m ²)	47 (22%)		81 (22%)		
Obese (≥30 kg/m ²)	71 (33%)		58 (16%)		
Second-hand smoke at home during current pregnancy					
(months)	224	3.14±3.76	391	1.50±2.99	
(h/day)	224	1.41±2.98	391	0.63±2.35	<0.01
Second-hand smoke at home in past 2 years					
(months)	224	8.43±10.65	391	3.76±8.16	<0.01
(h/day)	224	1.65±3.39	391	0.69±2.48	<0.01
No of cigarette smoked in presence of woman/day	224	2.95±6.60	391	1.48±4.89	<0.01
Currently married or cohabited ≥7 years (% yes)	35 (16%)		134 (34%)		<0.01
Frequent dietary PAH intake (% yes) [†]	75 (34%)		56 (15%)		<0.01
Maternal material hardship					
Could not pay for food	27 (12%)		77 (20%)		0.02
Could not pay for rent	30 (14%)		101 (26%)		<0.01
Could not pay for clothing	44 (20%)		131 (34%)		<0.01
Birth weight (cm)		3286±536		3419±456	<0.01
Fetal growth ratio		101.04±15.00		98.22±11.84	0.02
Birth length (cm)		50.62±2.95		50.82±2.53	0.42
Birth head circumference (cm)		33.85±1.76		34.37±1.36	<0.01
Gestational age (weeks)		39±2		39±1	<0.01
Parity (yes)	182 (81.3%)		280 (71.8%)		<0.01
Gender (female)	107 (50%)		202 (52%)		0.55

* Differences between African Americans and Dominicans were tested using the t test for continuous variables, in which equality of variance is not assumed and the χ^2 test for categorical variables.

[†] Consumed smoked, grilled or barbecued meat or fish at least twice per week.

Table 2

Mean birth weight per quartile range of airborne PAH, stratified according to suspected effect modifiers

PAH quartile range (ng/m ³)	Dominicans				African Americans			
	Mean birth weight (g)		Mean birth weight (g)		Mean birth weight (g)		Mean birth weight (g)	
	N	(95% CI)	N	(95% CI)	N	(95% CI)	N	(95% CI)
Maternal obesity before pregnancy onset								
<1.55	Non-obese (reference)		Obese		Non-obese (reference)		Obese	
	71	3401 (3291 to 3510)	13	3351 (3151 to 3551)	24	3365 (3196 to 3534)	12	3378 (2913 to 3844)
	70	3426 (3321 to 3531)	15	3522 (3302 to 3742)	38	3384 (3244 to 3525)	13	3682 (3393 to 3971)
	68	3395 (3284 to 3507)	10	3578 (3226 to 3929)	43	3224 (3088 to 3359)	18	3530 (3266 to 3795)
3.66+	74	3372 (3276 to 3468)	11	3726 (3378 to 4075)	36	3043 (2863 to 3223)	19	3051 (2805 to 3297)
Marital status during present pregnancy								
<1.55	Unmarried		Married/cohabiting (reference)		Unmarried		Married/cohabiting (reference)	
	28	3309 (3126 to 3493)	56	3435 (3322 to 3548)	7	3552 (3105 to 3999)	29	3325 (3118 to 3532)
	33	3500 (3384 to 3616)	52	3407 (3271 to 3543)	11	3577 (3348 to 3807)	40	3428 (3273 to 3583)
	25	3395 (3244 to 3545)	53	3430 (3289 to 3572)	4	3016* (2480 to 3551)	57	3335 (3205 to 3465)
3.66+	31	3422 (3257 to 3586)	54	3415 (3293 to 3537)	11	3072 (2742 to 3402)	44	3039 (2877 to 3201)
Second-hand smoke exposure ≥2 h/day at home								
<1.55	No (reference)		Yes		No (reference)		Yes	
	75	3386 (3283 to 3489)	9	3455 (3151 to 3759)	30	3389 (3170 to 3608)	6	3270 (2987 to 3553)
	75	3466 (3366 to 3565)	10	3272 (2982 to 3562)	44	3441 (3292 to 3590)	7	3579 (3373 to 3784)
	74	3431 (3320 to 3542)	4	3195 (2812 to 3578)	47	3292 (3160 to 3424)	14	3387 (3047 to 3728)
3.66+	77	3416 (3311 to 3521)	8	3431 (3193 to 3670)	36	3055 (2870 to 3240)	19	3028* (2795 to 3262)
Dietary intake of PAH								
<1.55	Low (reference)		High		Low (reference)		High	
	64	3424 (3320 to 3528)	15	3318 (3053 to 3582)	25	3406 (3175 to 3636)	10	3319 (2959 to 3678)
	69	3432 (3331 to 3532)	12	3500 (3173 to 3826)	36	3465 (3290 to 3641)	13	3420 (3244 to 3596)
	64	3380 (3264 to 3495)	12	3712 (3456 to 3967)	39	3293 (3132 to 3455)	21	3334 (3116 to 3552)
3.66+	66	3391 (3288 to 3493)	10	3489 (3192 to 3785)	32	3086 (2877 to 3294)	22	3007* (2812 to 3202)
Parity								
Primiparous			Multiparous (reference)			Primiparous		
						Multiparous (reference)		

PAH quartile range (ng/m ³)	Dominicans				African Americans			
	Mean birth weight (g)		Mean birth weight (g)		Mean birth weight (g)		Mean birth weight (g)	
	N	(95% CI)	N	(95% CI)	N	(95% CI)	N	(95% CI)
<1.55	20	3239 (2962 to 3516)	64	3441 (3349 to 3534)	9	3237 (2908 to 3566)	27	3414 (3188 to 3639)
1.55–2.29	31	3541 (3392 to 3690)	53	3372 (3252 to 3491)	15	3362 (3128 to 3596)	36	3501 (3342 to 3660)
2.30–3.65	21	3348 (3164 to 3532)	57	3445 (3314 to 3575)	7	3184 (2822 to 3546)	54	3331 (3195 to 3467)
3.66+	23	3395 (3229 to 3560)	62	3426 (3307 to 3546)	8	2948 (2542 to 3353)	47	3063 (2907 to 3218)
Highest maternal education attainment								
	≥High school (reference)		<High school		≥High school (reference)		<High school	
<1.55	27	3387 (3212 to 3563)	15	3165 (2928 to 3402)	21	3307 (3024 to 3590)	10	3590 (3367 to 3812)
1.55–2.29	31	3515 (3394 to 3636)	14	3452 (3182 to 3723)	32	3484 (3316 to 3651)	9	3298 (3012 to 3583)
2.30–3.65	41	3439 (3288 to 3589)	16	3468 (3226 to 3709)	32	3378 (3215 to 3540)	16	3267 (3016 to 3518)
3.66+	41	3438 (3286 to 3590)	26	3522 (3380 to 3663)	28	3100 (2917 to 3282)	18	2965 (2650 to 3279)
Maternal hardship during present pregnancy								
	None (reference)		≥One domain		None (reference)		≥One domain	
<1.55	54	3384 (3273 to 3495)	30	3410 (3223 to 3597)	27	3390 (3153 to 3626)	9	3309 (3048 to 3570)
1.55–2.29	40	3487 (3350 to 3624)	45	3404 (3272 to 3536)	41	3440 (3286 to 3594)	10	3544 (3308 to 3779)
2.30–3.65	46	3502 (3374 to 3630)	32	3300 (3119 to 3480)	40	3321 (3171 to 3471)	21	3301 (3061 to 3541)
3.66+	44	3365 (3229 to 3500)	41	3475 (3335 to 3614)	36	3044 (2859 to 3228)	19	3050 (2815 to 3285)

Separate analysis was conducted for each ethnic group.

* Significantly lower birth weight ($p<0.05$) compared with the African-American newborns at the highest quartile range without the corresponding traits.

PAH, polycyclic aromatic hydrocarbon.

Table 3

Comprehensive forward selection model of birth weight reduction in the overall cohort

	Model 1 [*]			Model 2 [*]			Model 3 [*]		
Adjusted-R ²	g Change	(95% CI)	p Value	g Change	(95% CI)	p Value	g Change	(95% CI)	p Value
(Constant)	3367	(3235 to 499)	0.000	3395	(3262 to 527)	0.000	3352	(3214 to 3489)	0.000
Main exposure									
Airborne PAH [†] × ethnic identity [‡]	-164	(-277 to -51)	0.005	-49	(-180 to 82)	0.459	-57	(-190 to 75)	0.395
Covariates									
Airborne PAH [†]	54	(-10 to 118)	0.096	25	(-41 to 92)	0.453	32	(-37 to 101)	0.361
Ethnic identity [‡]	86	(-45 to 218)	0.197	-40	(-191 to 111)	0.602	8	(-152 to 168)	0.920
Centered gestational age	81	(44 to 118)	0.000	79	(42 to 115)	0.000	78	(41 to 114)	0.000
Squared (centered gestational age)	-19	(-27 to -11)	0.000	-21	(-29 to -12)	0.000	-21	(-30 to -13)	0.000
Newborn gender [§]	-111	(-185 to -36)	0.004	-113	(-187 to -39)	0.003	-115	(-190 to -40)	0.003
Parity	58	(-27 to 144)	0.179	51	(-34 to 136)	0.237	45	(-42 to 132)	0.308
Delivery during spring	-125	(-209 to -40)	0.004	-134	(-218 to -50)	0.002	-126	(-211 to -41)	0.004
Second-hand smoke ≥2 h/day	-102	(-209 to 6)	0.064	-103	(-210 to 3)	0.058	-110	(-218 to -2)	0.047
Weight gain during pregnancy	9	(3 to 15)	0.004	10	(4 to 16)	0.001	12	(6 to 18)	0.000
Prepregnancy obesity [¶]	141	(45 to 236)	0.004	-122	(-341 to 97)	0.274	-139	(-368 to 90)	0.233
Frequent dietary PAH intake ^{**}							122	(-9 to 253)	0.068
Effect modification of airborne PAH									
Obesity × airborne PAH [†]				263	(59 to 468)	0.012	256	(40 to 471)	0.020
Obesity × ethnic identity [‡]				567	(248 to 886)	0.001	573	(247 to 899)	0.001
Obesity × airborne PAH × ethnic identity [‡]				-533	(-820 to -47)	0.000	-491	(-786 to -197)	0.001
Effect modification for African-American									
Dietary PAH ^{**} × ethnic identity [‡]							-204	(-387 to -21)	0.029

^{*} Dependent variable is birth weight.[†] Unit exposure was one natural log (ln) increase in transformed summed eight carcinogenic polycyclic aromatic hydrocarbons (PAH).[‡] African American=1, Dominican=0 (reference group).

§ Girl=1; boy=0 (reference group).

¶ Prepregnancy body mass index ≥ 30 .

** Consume at least one of the smoked, grilled, barbequed items more than twice a week.